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NEWS	17	JUN 25	CA/CAPLUS and USPAT databases updated with IPC reclassification data
NEWS	18	JUN 30	AEROSPACE enhanced with more than 1 million U.S. patent records
NEWS	19	JUN 30	EMBASE, EMBAL, and LEMBASE updated with additional options to display authors and affiliated organizations
NEWS	20	JUN 30	STN on the Web enhanced with new STN AnaVist Assistant and BLAST plug-in
NEWS	21	JUN 30	STN AnaVist enhanced with database content from EPFULL
NEWS	22	JUL 28	CA/CAPLUS patent coverage enhanced
NEWS	23	JUL 28	EPFULL enhanced with additional legal status information from the EPOLINE Register
NEWS	24	JUL 28	IFICDB, IFIPAT, and IFIUDB reloaded with enhancements
NEWS	25	JUL 28	STN Viewer performance improved
NEWS	26	AUG 01	INPADOCDB and INPAFAMDB coverage enhanced

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=> S (raf-1 (3A) RBD) (8A) (variant or mutant or mutated or mutation or mutating or mutagenesis or substitution or substitute or substituted)

L1            2 (RAF-1 (3A) RBD) (8A) (VARIANT OR MUTANT OR MUTATED OR MUTATION OR MUTATING OR MUTAGENESIS OR SUBSTITUTION OR SUBSTITUTE OR SUBSTITUTED)

=> S (gtpas or ras) (8A) (binding affinity)

L2            66 (GTPAS OR RAS) (8A) (BINDING AFFINITY)

=> s l1 and l2

L3            0 L1 AND L2

=> d l1 1-2 bib ab

L1    ANSWER 1 OF 2    EMBASE    COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN

AN    1999061407    EMBASE

TI    Nuclear magnetic resonance and molecular dynamics studies on the interactions of the Ras-binding domain of Raf-1 with wild-type and mutant Ras proteins.

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 SO Journal of Molecular Biology, (12 Feb 1999) Vol. 286, No. 1, pp. 219-232.  
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 AB The Ras protein and its homolog, Rap1A, have an identical 'effector  
 region' (residues 32-40) preceded by Asp30-Glu31 and Glu30-Lys31,  
 respectively. In the complex of the 'Ras-like' E30D/K31E mutant Rap1A  
 with the Ras-binding domain (RBD), residues 51-131 of Raf-1, Glu31 in  
 Rap1A forms a tight salt bridge with Lys84 in Raf-1. However, we have  
 recently found that Raf-1 RBD binding of Ras is indeed reduced by the E31K  
 mutation, but is not affected by the E31A mutation. Here, the  
 'Rap1A-like' D30E/E31K mutant of Ras was prepared and shown to bind the  
 Raf-1 RBD less strongly than wild-type Ras, but slightly more tightly than  
 the E31K mutant. The backbone (1)H, (13)C, and (15)N magnetic resonances  
 of the Raf-1 RBD were assigned in complexes with the wild-type and  
 D30E/E31K mutant Ras proteins in the guanosine 5'-O-( $\beta,\gamma$ -  
 imidotriphosphate)-bound form. The Lys84 residue in the Raf-1 RBD  
 exhibited a large change in chemical shift upon binding wild-type Ras,  
 suggesting that Lys84 interacts with wild-type Ras. The D30E/E31K mutant  
 of Ras caused nearly the same perturbations in Raf-1 chemical shifts,  
 including that of Lys84. We hypothesized that Glu31 in Ras may not be the  
 major salt bridge partner of Lys84 in Raf-1. A molecular dynamics  
 simulation of a model structure of the Raf-1 RBD.ovrhdot.Ras.ovrhdot.GTP  
 complex suggested that Lys84 in Raf-1 might instead form a tight salt  
 bridge with Asp33 in Ras. Consistent with this, the D33A mutation  
 in Ras greatly reduced its Raf-1 RBD binding  
 activity. We conclude that the major salt bridge partner of Lys84 in  
 Raf-1 may be Asp33 in Ras.

L1 ANSWER 2 OF 2 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN  
 AN 1999:134549 BIOSIS  
 DN PREV199900134549  
 TI Nuclear magnetic resonance and molecular dynamics studies on the  
 interactions of the Ras-binding domain of Raf-1 with wild-type and mutant  
 Ras proteins.  
 AU Terada, Tohru; Ito, Yutaka; Shirouzu, Mikako; Tateno, Masaru; Hashimoto,  
 Kyoko; Kigawa, Takanori; Ebisuzaki, Toshikazu; Takio, Koji; Shibata,  
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 SO Journal of Molecular Biology, (Feb. 12, 1999) Vol. 286, No. 1, pp.  
 219-232. print.

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DT Article

LA English

ED Entered STN: 31 Mar 1999

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